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NEWS 1 Web Page for STN Seminar Schedule - N. America  
NEWS 2 AUG 10 Time limit for inactive STN sessions doubles to 40  
minutes  
NEWS 3 AUG 18 COMPENDEX indexing changed for the Corporate Source  
(CS) field  
NEWS 4 AUG 24 ENCOMPLIT/ENCOMPLIT2 reloaded and enhanced  
NEWS 5 AUG 24 CA/CAPLUS enhanced with legal status information for  
U.S. patents  
NEWS 6 SEP 09 50 Millionth Unique Chemical Substance Recorded in  
CAS REGISTRY  
NEWS 7 SEP 11 WPIDS, WPINDEX, and WPIX now include Japanese FTERM  
thesaurus  
NEWS 8 OCT 21 Derwent World Patents Index Coverage of Indian and  
Taiwanese Content Expanded  
NEWS 9 OCT 21 Derwent World Patents Index enhanced with human  
translated claims for Chinese Applications and  
Utility Models  
NEWS 10 OCT 27 Free display of legal status information in CA/CAPLUS,  
USPATFULL, and USPAT2 in the month of November.

NEWS EXPRESS MAY 26 09 CURRENT WINDOWS VERSION IS V8.4,  
AND CURRENT DISCOVER FILE IS DATED 06 APRIL 2009.

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FILE 'HOME' ENTERED AT 11:21:01 ON 09 NOV 2009

=> file medline biosis caplus embase

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FULL ESTIMATED COST	0.44	0.44

FILE 'MEDLINE' ENTERED AT 11:22:01 ON 09 NOV 2009

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```
=> s insulin(w)receptor(w)substrate(w)2 or IRS(w)2 and (activator or inhibitor)
L1      3541 INSULIN(W) RECEPTOR(W) SUBSTRATE(W) 2 OR IRS(W) 2 AND (ACTIVATOR
      OR INHIBITOR)
```

```
=> s l1 and (over(w)express or over(w)produce)
L2      2 L1 AND (OVER(W) EXPRESS OR OVER(W) PRODUCE)
```

```
=> dup rem l2
PROCESSING COMPLETED FOR L2
L3      2 DUP REM L2 (0 DUPLICATES REMOVED)
```

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=> dis ibib abs l3
```

L3 ANSWER 1 OF 2 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2005199769 EMBASE

TITLE: The GLUTs family - Lessons from transgenic mice.

AUTHOR: Hartil, K.; Weldon, R.H.; Seki, Y.; Charron, M.J.  
(correspondence)

CORPORATE SOURCE: Department of Biochemistry, Albert Einstein College of Medicine, 1300 Morris Park Avenue, Bronx, NY 10461, United States. charron@aeacom.yu.edu

SOURCE: Current Medicinal Chemistry: Immunology, Endocrine and Metabolic Agents, (Apr 2005) Vol. 5, No. 2, pp. 189-206.  
Refs: 144

ISSN: 1568-0134 CODEN: CMCIC8

COUNTRY: Netherlands

DOCUMENT TYPE: Journal; General Review; (Review)

FILE SEGMENT: 016 Cancer  
018 Cardiovascular Diseases and Cardiovascular Surgery  
022 Human Genetics  
029 Clinical and Experimental Biochemistry  
003 Endocrinology  
005 General Pathology and Pathological Anatomy

LANGUAGE: English

SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 19 May 2005

Last Updated on STN: 19 May 2005

AB The glucose transporters (GLUTs) are currently a 13 member family of facilitative transmembrane proteins which transport glucose down its concentration gradient. The GLUTs have a tissue specific expression and regulation. Dysregulation of GLUTs have been implicated in the pathogenesis of a number of diseases including diabetes and cancer and are known to play an important role in the developing embryo. In addition, roles for GLUTs in cardiac function and embryonic development have been identified and will be discussed in this review. The ability to ablate or over-express GLUTs has advanced our understanding of the role these transporters play in the maintenance of normal glucose homeostasis and the pathogenesis of diabetes. The development of Cre-LoxP technology coupled with the existence of tissue specific promoters allows

investigators to manipulate gene expression both globally and in a tissue specific manner. The major GLUTs which have been investigated using transgenic technology are GLUT1, GLUT4 and GLUT2. Overexpression of GLUT4 and GLUT1 results in increased glucose uptake and metabolism. However, only GLUT4 overexpression protects against the development of insulin resistance in transgenic mice. Genetic ablation of GLUT4 and GLUT2 results in impaired insulin tolerance and defects in both lipid and glucose metabolism. This review will present various transgenic models of GLUT modification and discuss what has been learned from these models about the role that GLUTs play in glucose homeostasis, insulin action and development. .COPYRG. 2005 Bentham Science Publishers Ltd.

=> dis ibib abs 13 2

L3 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2009 ACS on STN  
ACCESSION NUMBER: 2001:129407 CAPLUS  
DOCUMENT NUMBER: 134:261420  
TITLE: Specific inhibition by hGrb10 $\zeta$  of insulin-induced glycogen synthase activation: evidence for a novel signaling pathway  
AUTHOR(S): Mounier, C.; Lavoie, L.; Dumas, V.; Mohammad-Ali, K.; Wu, J.; Nantel, A.; Bergeron, J. J. M.; Thomas, D. Y.; Posner, B. I.  
CORPORATE SOURCE: The Polypeptide Hormone Laboratory, McGill University, Montreal, QC, H3A 2B2, Can.  
SOURCE: Molecular and Cellular Endocrinology (2001), 173(1-2), 15-27  
CODEN: MCEND6; ISSN: 0303-7207  
PUBLISHER: Elsevier Science Ireland Ltd.  
DOCUMENT TYPE: Journal  
LANGUAGE: English  
AB Grb10 is a member of a family of adapter proteins that binds to tyrosine-phosphorylated receptors including the insulin receptor kinase (IRK). In this study recombinant adenovirus was used to over-express hGrb10 $\zeta$ , a new Grb10 isoform, in primary rat hepatocytes and the consequences for insulin signaling were evaluated. Over-expression of hGrb10 $\zeta$  resulted in 50% inhibition of insulin-stimulated IRK autophosphorylation and activation. Anal. of downstream events showed that hGrb10 $\zeta$  over-expression specifically inhibits insulin-stimulated glycogen synthase (GS) activity and glycogen synthesis without affecting insulin-induced IRS1/2 phosphorylation, PI3-kinase activation, insulin like growth factor binding protein-1 (IGFBP-1) mRNA expression, and ERK1/2 MAP kinase activity. The classical pathway from PI3-kinase through Akt-PKB/GSK-3 leading to GS activation by insulin was also not affected by hGrb10 $\zeta$  over-expression. These results indicate that hGrb10 $\zeta$  inhibits a novel and presently unidentified insulin signaling pathway leading to GS activation in liver.  
OS.CITING REF COUNT: 27 THERE ARE 27 CAPLUS RECORDS THAT CITE THIS RECORD (27 CITINGS)  
REFERENCE COUNT: 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

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ALL L# QUERIES AND ANSWER SETS ARE DELETED AT LOGOFF  
LOGOFF? (Y)/N/HOLD:y

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L1 FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE' ENTERED AT 11:22:01 ON 09 NOV 2009  
3541 SEA FILE=MFE SPE=ON ABB=ON PLU=ON INSULIN(W) RECEPTOR(W)

SUBSTRATE(W) 2 OR IRS(W) 2 AND (ACTIVATOR OR INHIBITOR)  
L2                  2 SEA FILE=MFE SPE=ON ABB=ON PLU=ON L1 AND (OVER(W) EXPRESS  
                          OR OVER(W) PRODUCE)  
L3                  2 DUP REM L2 (0 DUPLICATES REMOVED)  
                          DIS IBIB ABS L3  
                          DIS IBIB ABS L3 2

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	ENTRY	SESSION
FULL ESTIMATED COST	40.11	40.55
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE	TOTAL
	ENTRY	SESSION
CA SUBSCRIBER PRICE	-0.82	-0.82

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